proximity of the hepatic flexure of the colon to the liver strongly suggest that decompression occurred through the colon. The otherwise uncomplicated recovery of this patient shows the possibly benign natural course of an amebic liver abscess that drains into the colon.

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Neisseria sicca Endocarditis in Intravenous Drug Abusers

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Neisseria sicca is one of many species of *Neisseria* that are normal inhabitants of the nasopharynx. This organism has generally been considered nonpathogenic. Since 1918 there have been seven cases of *N sicca* endocarditis reported in the literature.[1] We now report two additional cases in intravenous drug abusers.

Reports of Cases

Case 1

The patient, a 41-year-old man with a history of intravenous abuse of codeine and methylphenidate hydrochloride, was admitted to Los Angeles County-University of Southern California (LAC-USC) Medical Center because of malaise, fever and chills starting four days before admission. He had sporadically taken erythromycin and tetracycline orally for exhaustion. On physical examination at admission, he had an oral temperature of 40.5°C (104.9°F), many dental caries and a grade 1/6 systolic ejection murmur, best heard along the left sternal border. His peripheral leukocyte count was 15,600 per μl, with 80% polymorphonuclear leukocytes and 4% band cells. A chest radiograph showed infiltrates in the left midlung and right lower lung fields, consistent with embolization. Infective endocarditis was suspected. Blood specimens were drawn for culture and the patient was given vancomycin hydrochloride intravenously (he had a history of penicillin allergy) and gentamicin sulfate. Six days later, one of five of the blood specimens cultured yielded Gram-negative cocci, later identified as *N sicca*. The patient's fever resolved, but on the seventh hospital day the central venous catheter was accidentally disconnected. Because all but one blood culture were negative for the pathogen, it was decided not to reinstate the intravenous line; instead, the patient was given erythromycin by mouth. Fever of 40.5°C (104.9°F) subsequently developed. Treatment with vancomycin and gentamicin was begun again, but high fever continued. On the ninth day in hospital, his treatment was changed to intravenous administration of cefazolin sodium, resulting in defervescence after two days.

On the 13th day the patient was transferred to Rancho Los Amigos Hospital (Downey, Calif) for continuation of antibiotic therapy for a presumptive culture-negative infective endocarditis. The antibiotic regimen was discontinued for two days to determine the true nature of his bacteremia. Within 24 hours his temperature rose to 41.6°C (107°F). He was consequently treated again with vancomycin and gentamicin. Six of seven sets of cultures of blood specimens obtained during the time when the patient was not receiving antibiotics yielded *N sicca* sensitive to penicillin. The patient left the hospital against medical advice before therapy was completed.

Case 2

The patient, a 24-year-old man with a history of intravenous cocaine and heroin abuse, was admitted to LAC/USC Medical Center after experiencing fever, chills, malaise, right pleuritic pain and transient hemoptysis for ten days. The fever and chills had persisted despite one week of self-treatment with penicillin taken orally and ampicillin. He had had no previous cardiac disease or heart murmurs. On admission the patient had a temperature of 40°C (104°F) accompanied by shaking chills. Physical examination was remarkable for a grade 3/6 early systolic murmur at the left midsternal border, which was accentuated by inspiration or squatting. He had a leukocyte count of 9,500 per μl with 84% polymorphonuclear leukocytes and 3% band cells. A chest radiograph showed nodular infiltrates in the right lower lobe. The patient was given oxacillin sodium intravenously and tobramycin for a tentative diagnosis of bacterial endocarditis with multiple septic pulmonary emboli. Six out of six blood cultures from specimens taken on admission yielded oxacillin-sensitive *Staphylococcus aureus*. Treatment with tobramycin was discontinued. An echocardiogram showed a "pea-sized" tricuspid vegetation. The patient's fever subsided and serial chest films showed gradual resolution of the infiltrates. After ten days of oxacillin therapy, the patient left the hospital against medical advice.

He returned to the medical center 12 days after his initial admission with a temperature of 40°C (104°F), shaking chills and an unchanged heart murmur. The patient was given oxacillin intravenously after three specimens of blood for culture were drawn. The blood cultures yielded no growth.

The patient was transferred to Rancho Los Amigos Hospital 17 days after his readmission to complete a four-week course of parenteral antibiotic therapy. He was treated with nafcillin sodium given intravenously. One day after comple-

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tion of his four-week course of antibiotic therapy, fever to 39°C (102.2°F) ensued. Nafcillin therapy was restarted after blood was drawn for culture. His therapy was changed to intravenous administration of ampicillin when ampicillin-sensitive *N sicca* was isolated from six of eight sets of blood cultures. The patient responded well to ampicillin therapy and his fever dropped for several days, after which left pleuritic pain and low-grade fever developed. A chest roentgenogram and ventilation-perfusion scan were suggestive of new pulmonary emboli at the left lower lobe; these gradually resolved in a week. Ampicillin therapy was discontinued after completion of a four-week course. The patient remained afebrile for ten days, at which time fever and shaking chills recurred. Ampicillin-sensitive *N sicca* was once again isolated from three of four sets of blood cultures. Removal of the tricuspid valve was contemplated and the patient was transferred to Harbor-UCLA Medical Center (Torrance, Calif).

At Harbor-UCLA Medical Center, the patient was given penicillin and gentamicin, but fever persisted and bilateral lower lobe infiltrates developed. Oxacillin was added to the regimen of penicillin and gentamicin. The patient's fever gradually resolved and he completed four weeks of therapy with these antibiotics. Blood cultures yielded no growth after the antibiotic regimen was discontinued and the patient remained afebrile until discharge to home.

Microbiology

Blood specimens for culture were collected in 90-ml bottles of trypticase soy broth and Schaedler's medium, each containing 0.03% sodium polyanethol-sulfonate and 6% carbon dioxide (BBL, Cockeysville, Md). The trypticase soy broth was vented before incubation of the cultures. The organisms were positive for oxidase and showed acid reaction in cysteine trypticase agar for glucose, maltose, fructose and sucrose but not lactose. Microtube-dilution susceptibility testing[3] showed that both organisms were susceptible to penicillin, ampicillin, chloramphenicol, tetracycline, methicillin and gentamicin. Macrotube-dilution susceptibility testing for ampicillin and gentamicin showed minimal inhibitory concentrations of 0.4 and 0.8 μg per ml for the isolate from patient 1 and 0.2 and 0.02 μg per ml for isolate from patient 2. Minimal bactericidal concentrations for ampicillin and gentamicin were 0.4 and 0.8 μg per ml for the first patient and 0.4 and 0.05 μg per ml for the second. A synergistic isobologram study for ampicillin and gentamicin was done on isolate No. 2 and showed antagonism between the two drugs (Figure 1). A test for β-lactamase production on this same isolate, using the chromogenic cephalosporin (Cefinase, BBL) disc method yielded a negative result. Serum bactericidal activity in patient 2 occurred at a 1:32 dilution.

Discussion

Patients with *N sicca* endocarditis in cases reported in the literature presented with symptoms similar to those seen in these two men. They had fever with temperatures as high as 39°C (102°F) or more, sudden onset of symptoms, multiple embolic phenomena and delayed arrival at the diagnosis. Mortality was extremely high in the preantibiotic era.[1] The first successfully treated case was reported in 1941 when the patient described received three weeks of heparin and sulfapyridine therapy.[2] The remaining patients were treated with

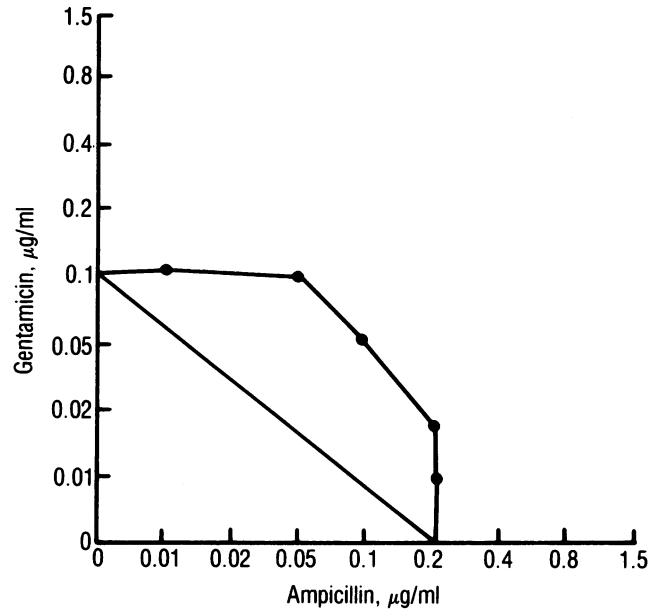

Figure 1.—Isobologram of the minimal inhibitory concentrations of ampicillin and gentamicin for isolate No. 2.

either sulfapyridine or penicillin with or without streptomycin. The organisms found in the cases presented here were quite sensitive to penicillin and ampicillin.

The reason for the repeated medical failures in case 2 is unclear. Some possibilities should be considered: (1) inadequate response to ampicillin when used alone (in vitro studies, however, showed antagonism between ampicillin and gentamicin), (2) β-lactamase production that was neutralized by oxacillin, allowing ampicillin to work more effectively (however, this organism was β-lactamase-negative), (3) *S aureus* was not eradicated by previous therapy.

It is not clear why *N sicca* endocarditis develops in some persons. Poor dental hygiene and practices of licking and sharing of needles may be factors that predispose these patients to infection with bacteria from oral flora. It is difficult to say whether the fact that both of these patients were taking oral antibiotics while continuing to use drugs intravenously played any role in the development of their infection. The oral antibiotics may have suppressed the more exquisitely antibiotic-susceptible component of the oral flora, allowing the more resistant *N sicca* to become predominant and increasing their suceptibility to be infected by this "nonpathogenic" organism. *N sicca* should not be readily dismissed as a blood culture contaminant, especially in the setting of parenteral drug abuse, and should be added to the list of microorganisms responsible for infective endocarditis in intravenous drug abusers.

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